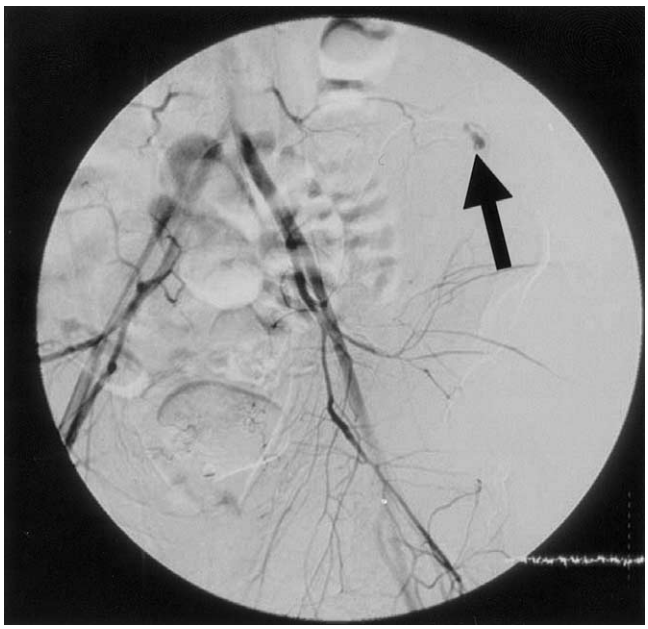

FIGURE 3. Arteriogram before embolization showing extravasation of contrast media from the branch of the left internal iliac artery (arrow).

For severe hemorrhagic shock, TABO provides an alternative to thoracotomy with aortic crossclamping.[4,5] TABO before a curable hemostatic procedure is effective, and a 9-Fr balloon catheter (Block balloon; AISIN SEIKI Co., Ltd, Kariya, Japan) for TABO through a 10-Fr sheath has been developed recently.[4,5] Although insertion of the introducer into the femoral artery could fail as a result of a decrease of blood pressure and succeeding arterial spasm in severe hypovolemic shock, the exchange of an 8-Fr introducer for a 10-Fr one using the Seldinger technique is easy.[4] Thus, we performed preoperative placement of the 8-Fr sheath in readiness for immediate intraoperative TABO. Although we have not performed TABO, we believe that we can have perform craniotomy with a low risk of deterioration of the hemodynamic condition.

In conclusion, EPs should consider the preceding placement of an arterial sheath in cases of multiple trauma with active bleeding, especially in cases that require radiographic embolization.

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FATAL UPPER AIRWAY OBSTRUCTION INDUCED BY SUPERIOR MEDIASTINUM BLEEDING

To the Editor:—Cervical hematoma is one of the causes of upper airway obstruction.[1-5] We report a case of fatal upper airway obstruction possibly caused by aortic arch rupture–induced cervical internal hemorrhage.

Sudden cardiac arrest occurred in a 63-year-old man while undergoing body massotherapy in the prone position. After short-term stridor, he lost consciousness. Rapid swelling of the neck was observed. Bystander cardiopulmonary resuscitation (CPR), including mouth-to-mouth ventilation and cardiac massage, was immediately started. Nine minutes later, life-saving technicians took over CPR. No obstructing substance was found in his oral space. Ventilation through an inserted combitube airway was impossible. Pulseless electric activity on an electrocardiogram (ECG) was confirmed.

At 22 minutes after the start of CPR, the patient arrived at our emergency room (Glasgow Coma Scale: 3, pulseless electric activity on ECG). Epinephrine (1 mg intravenously) increased the heart rate on ECG from 35 to 65/min but did not increase blood pressure. Because ventilation was still impossible, the combitube airway was removed. Subsequent laryngoscopy was extremely difficult; the supraglottic and glottic areas were filled with mucosal edema and blocked by an upheaved pharyngeal posterior wall, making discrimination of the epiglottis, arytenoids cartilage, and vocal cords impossible. In contrast, no significant edema was seen in oral, nasal, and conjunctival mucosae. Immediate cricothyrotomy was executed, and control ventilation then became possible with no respiratory deficiency (results of blood gas analysis: Pa_{O_2}, 87.1 mm Hg; Pa_{CO_2}, 27.7 mm Hg; pH, 6.925; base excess, −24.0 at 10 min after the resumption of ventilation). Intravenous administration of 150 mL of 7% $NaHCO_3$ and 2 mg epinephrine after resumption of ventilation improved circulation (arterial blood pressure, 66/42 mm Hg; heart rate, 141/min). Thereafter, dopamine (15 μg/kg/min) was intravenously administrated to maintain blood pressure.

Dilation of the upper mediastinum was seen in a radiograph (Fig 1). The patient's swollen neck was strained. Cervical echograms showed bilateral, irregular-shaped, low-echo areas with unclear outlines around the internal carotid arteries. Internal juglar veins, which should appear near the internal carotid arteries, could not be discriminated in echograms (Fig 2). Inspection with a bronchofiber showed apparently normal tracheal mucosa and cartilage but a greatly upheaved tracheal membranous portion. No hemothorax was found in echograms or radiographs.

Neurologic recovery was not achieved. Dopamine administration failed to maintain blood pressure and heart rate, and the circulation thus gradually deteriorated. The patient died about 115 minutes after the start of CPR. A postmortem inquest using a

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laryngofiber revealed that the mucosal edema and upheaval of the posterior wall in the pharynx had been reduced but still remained.

Diagnosis of the cause of upper-airway obstruction in this case while executing CPR was difficult. Allergic reaction was not thought to have been the cause because oral, nasal, conjunctival, and tracheal mucosae were not edematous and no other allergic symptoms were found. We suspected cervical space occupying lesions (SOLs) shown by the bilaterally expanded cervical low-echo areas to be the cause of the airway obstruction. Compression of the bilateral internal juglar veins by the SOLs might have caused deterioration in local circulation and then elicited the severe mucosal edema, and the SOLs might also have directly caused upheaval of the pharyngeal posterior wall. The synchronized onset of respiratory distress and cervical swelling supports this hypothesis regarding the cause. The cervical swelling was rapid and seemed to be increasing tissue pressure. The compressed internal juglar veins, upheaved pharyngeal posterior wall, and upheaved tracheal membrane portion indicate increase in pressure of cervical and peritracheal tissue. The upper mediastinum was dilated. These findings suggested that the SOL was most likely to be an arterial hemorrhage–induced hematoma spreading in the upper mediastinum and bilateral cervical area. We also considered tumor and inflammation as the possible contents of the SOLs; however, these could not explain the very rapid onset of upper-airway obstruction. Bilaterality of the cervical hematomas strongly suggests that a bleeding point did not exist on 1 side of the internal carotid arteries. We strongly suspect that the bleeding point existed in the dilated upper mediastinum, most likely at the top of the aortic arch where the carotid arteries originate. Acute upper-airway obstruction after accidental puncture of the internal carotid artery has been reported,[3-5] but, we have found no other reports of upper airway obstruction induced by superior mediastinum bleeding. Aortic arch rupture in the superior mediastinum should be regarded as one of the mechanisms eliciting acute upper-airway obstruction.

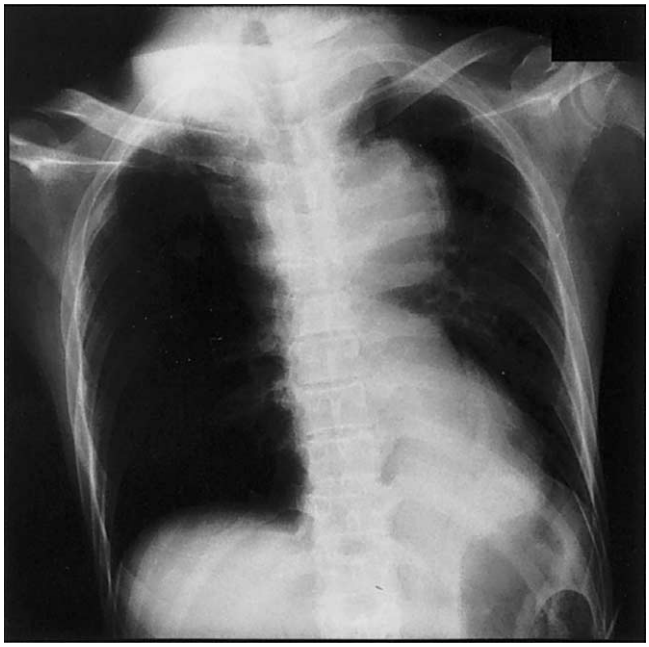

FIGURE 1. Chest radiograph showing dilated upper mediastinum.

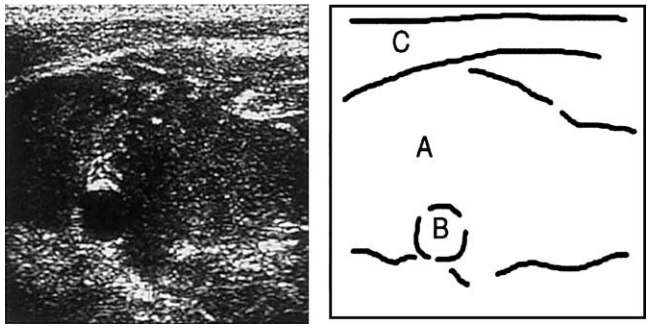

FIGURE 2. Cervical echogram showing low-echo area with an unclear outline around the right carotid artery. The right internal juglar vein, which should appear near the carotid artery, could not be discriminated. A, Low-echo area; B, internal carotid artery; C, sternocleidomastoid muscle.

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